



An overview of white rust disease in Brassica: taxonomical, biochemical aspects and management approaches

Muhammad Asif¹, Muhammad SaqibMushtaq¹, Hina Firdous¹, Muhammad Mubashar Zafar²✉, Ali Imran², Tanvir Ahmad¹, Hafiz Muhammad Arslan Abid¹, Hafiz Saad Bin Mustafa³

1.Department of Plant Pathology, University of Agriculture Faisalabad, Pakistan

2.Department of Plant Breeding and Genetics, University of Agriculture Faisalabad, Pakistan

3.Director of Oilseeds, Ayub Agricultural Research Institute Faisalabad, Pakistan

✉Corresponding author:

Department of Plant Breeding and Genetics,
University of Agriculture Faisalabad,
Pakistan
Email: m.mubasharzafar@gmail.com

Article History

Received: 23 August 2017

Accepted: 17 September 2017

Published: 1 November 2017

Citation

Muhammad Asif, Muhammad SaqibMushtaq, Hina Firdous, Muhammad Mubashar Zafar, Ali Imran, Tanvir Ahmad, Hafiz Muhammad Arslan Abid, Hafiz Saad Bin Mustafa. An overview of white rust disease in Brassica: taxonomical, biochemical aspects and management approaches. *Discovery*, 2017, 53(263), 571-586

Publication License



© The Author(s) 2017. Open Access. This article is licensed under a [Creative Commons Attribution License 4.0 \(CC BY 4.0\)](https://creativecommons.org/licenses/by/4.0/).

General Note



Article is recommended to print as color digital version in recycled paper.

ABSTRACT

Rapeseed (*Brassica napus* L.) belongs Brassicaceae family is the second most important oilseed crop in Pakistan after cotton. It contains 40-46% oil content and 22% protein. There are several factors which lowers seed yield like abiotic and biotic stresses. *Alternaria* blight and white rust diseases are the major threat for reducing Brassica seed yield in Pakistan. White Rust (WR) is the most destructive disease of brassica in tropical and subtropical areas of Pakistan caused by *Albugo candida*. It causes 20-90% yield losses throughout the world. In Pakistan, it was first reported from FATA and BAJAUR Agency with 32% pod losses and 52% foliage losses. *A. candida* is an obligate parasite and its survival rate is very high in plants debris and soil. It is reported from different Asian and European countries causing different epidemics. It has wide host range of *Cruciferae*, *Ficoidaceae*, *Cappaaracea*, and *Cleeeomaceae* white or off-white (creamy) raised pustules of pin size to various forms, and sizes leading to stag-head formation. Three types of spores are produced and their nature of infection is variable. Oospores are seed and air borne when carried on seed, or overwintered in soil; serve as prime source of infection in Brassica. Through extensive research different races of *A. candida* are identified on different host range. Few races like AC 2, 3, 4, 5, 6, 7, 8, 9 are common in various countries and different researcher named them accordingly. That's why it is being placed in different families, and genera. Infection of pathogen brings different biochemical changes in plant. These changes in host are useful to understand the biochemistry, host pathogen-interaction and to manage the disease by avoiding symptom's ambiguity. Screening of resistant germplasm is a prime factor and extensive research is being conducted in this regard. Resistant cultivars are the best option to control the disease as they are eco-friendly, locally adapted to the environment, and durable. Different genotypes of *Brassica juncea*, *B. carinata*, *B. napus* and *B. rapa* were evaluated in field conditions against white rust for five years. Resistant sources are mentioned in this review according to the local climate of different countries. Chemicals protect the plants by forming layer on leaves surface while some have ability to penetrate the system and help the plant to reverse the biochemical changes induced by the pathogen. The Symptomology of a disease plays an imperative role in the understanding of altered physiological changes and the establishment of pathogen with in host but there are many factors which caused the symptoms uttered by pathogenic fungus like infection time, type of infection, plant age, genetic makeup of the host plant and environmental conditions which need further research. Biochemical changes play an important role in the plant defense and induce resistance as the proteins form complex with fungus to inhibit it. One compound regulates the level of other one in tissues as H_2O_2 is produced at a very high rate in susceptible but less in moderately resistant lines. As a defensive action on H_2O_2 the quantity of CAT which led to increase in POD and SOD is increased to manipulate later and so is the way of regulating it. Catalase (CAT) prevents the accumulation of H_2O_2 in the cells. CAT activity increases in resistant varieties and it breaks down the hydrogen peroxide into water and oxygen. Peroxidase (POD) plays a significant role in defense response and its activity is associated with resistance induction in different species of plants. It is also involved in the breakdown of hydrogen peroxide. Increased activity of peroxidase leads to the production and accumulation of hydroxyproline rich glycoprotein into the cell wall. Peroxidase is involved in lignin polymerization. The enzyme has role in cell wall metabolism as well as in defense regulation and induction. Superoxidase dismutase enzyme acts as a first line of defense against reactive oxygen species (ROS) and rapid induction of SOD leads to recognize the pathogen's avirulence factors. phenolic contents can be increased due to glycosidic esters formed by the enzymatic activity of host or pathogen or due to migration of phenols from uninfected tissue. Our research findings support the above-mentioned facts. Different researcher has reported different chemicals of same formulations as Meralaxyl, Mancozeb, Captaf, Swing and Atracol to control white rust either using them as seed treatment or foliar application. These biochemical and chemicals interactions need more exploration.

Keywords: Antioxidant enzymes, Brassica, Racial variation, Management, White rust

1. INTRODUCTION

Rapeseed & Mustard are the third important edible oil producing crops in the world after soybean and palm oil. These crops grow well under both irrigated and rain-fed conditions and gives good seed yield (Mustafa et al., 2017). *Brassica napus* (L) belongs to family Brassicaceae and cultivated since 2000 B.C having 350 genera and 3700 species worldwide while 92 genera and 250 species are reported in Pakistan (Hina et al., 2014). Some well-known species of Rapeseed are *Brassica oleraceae*, *Brassic napus*, *Brassica rapa*, *Armoracia rusticana* P. Gaertn etc. Brassica is also named as Canola and its spp. like *Brassica. napus* and *Brassica juncea* are the conventional source of oil and have great importance as a food ingredient in the whole world especially in South Asia (Mishra et al., 2009). It was originated in South West Europe and Mediterranean coastal area and evolved by continuous crossing between *B. rapa*

and *B. oleracea* (Warwick et al., 2006; Shahzadi et al. 2015). Through various sources, it spread to Scandinavia, Western Asia, Japan, India, Finland, Canada, Sweden, and Pakistan as an oilseed crop. It is introduced through Canada in Pakistan (1995). It is cultivated in different Punjab Divisions like Islamabad, Rawalpindi, Attock, Lahore, Faisalabad, and Multan during mid-October to mid-November (Przybylski and Mag, 2002).

It is herbaceous annual plant having a short height of 45-150 cm. The roots are confined to rhizosphere with an extensive lateral spread. The stem is usually covered with a waxy deposit. Fruits are thicker than those of mustard plant. Self-pollination is commonly present but cross pollination rarely happens. It contains 40-46 % oil content and 18-22 % protein. Lysine, Cystine, and Methionine are found but such amino acids are mostly deficient in cereal meal (Rashid et al., 2005). Erucic acid 40-46%, glucosinolate content (80-160 μMg^{-1}), (Przybylski and Mag, 2002) linolenic acid 4.7-13% and oleic acid 27% is present in young leaves of in Indian and Pakistani cultivars. Linoleic acid is highly nutritious and valuable for human health (Kumar et al., 2014). Low Erucic Acid and glucosinolate are characteristic features of *Brassica napus* as safe limit of these compounds in oil meal should be less than 5%. Hybrids of Brassica have high yielding potential (Chaudry et al., 2011).

Brassica is grown in temperate climate at higher elevations. The optimum temperature required for growth and development is 18-25°C with low humidity. The maximum growth rate is observed at 25°C but this rate is slowed down at 35°C and 3°C respectively. Heavy rainfall, high humidity, and cloudy weather are unfavorable for the crop during germination in winter while under rainfed conditions if it is raining once or twice at the pre-flowering stage, it will be helpful in boosting the grain yield. Aphid can enter easily when there is high rainfall and damage the crop foliage. Cold and frost have the bad impact on foliage. So, optimum relative humidity (RH) must be 30-60% and annual precipitation 40-100cm. Brassica is intolerant to water-logging. Its growth is optimum in medium or heavy loam soils. The soil pH less than 5.0 is best suitable but pH 9.0 is detrimental for *Brassica* while soil with pH of 6.0-7.5 is ideal for the proper growth and development (Chattopadhyay et al., 2005).

2. HISTORICAL PERSPECTIVE OF DISEASE

White Rust (WR) is the common damaging disease of brassica in tropical and subtropical areas of Pakistan caused by *Albugo candida*. It is an obligate parasite (Armstrong, 2007). White rust (WR) is commonly called due to its white lesion or pustules development that is visible on the downside of infected leaf. It is also called as white rust of crucifers, stag-heads disease, and white blister rust. It is reported from different Countries like USA, Canada, South-America, Africa, Hong Kong, Texas, Brazil, Germany, India, Japan, Pakistan, Palestine, Romania, Turkey, Fiji, New Zealand, China, Korea (Choi et al., 2011a), Russia, Uruguay, Argentina and U.K. (Meena et al., 2014).

Albugo candida (AC) is identified from *Raphanus raphanistrum*, *Brassica napus* (cv "Rapa" and Nabrassica), Radish, Cabbage (wild), Mustard (Saharan), Chinese cabbage, Japanese mustard on Lucern (1940-1999) from Western Australia, California and United States of America (Koike, 1996). It causes huge yield losses of 1-60% in turnip rape (*Brassica rapa* L.) in Canada, 23-90% in Indian mustard (*Brassica juncea*) and 5-20% in Australia (Meena et al., 2014). Different reports indicated that infected Brassica crop will result in 47% less pods production and 33% less seed affecting the overall yield (Meena et al., 2014).

3. HOST RANGE

A. candida (AC) has a wide host range in Brassicaceae. There is a high degree of genomic versatility in this genus as many of the investigated lines possess a definite separate species. After the Lecto-typification analysis of *A. candida*, two new specific species (phyto-parasitic to host) are added in this genus (Choi et al., 2008). *Cleomaceae* and *Capparidaceae* are mostly infected by the pathogen (Choi et al., 2011.b). It can infect many Crucifers, Ficoidaceae, *Cappareaoracea*, and *Cleaeomaceae* and specifically infected host species are mignonette, kale, brown mustard, mustard black, Chinese cabbage, *Brassica pekinensis*, cauliflower, radish, horse dish, cress, water cress, *Cheiranthus cheiri*, *Matthiola incana*, *Raphanus raphanistrum* and several such weeds (Choi et al., 2006; Voglmayr and Riethmüller, 2006). Most common weed host are *Capsella shepard*, virgine pepper cress and hedge mustard (Kaur et al., 2011).

Due to symptomless infections, it travels upward through seeds so that fungus can develop relationships among crucifer plant hosts like *campestre*, *Arabis lyrata* L. and *Erysimum* mostly grown within Mediterranean region for edible flower buds. These species are also attacked by *A. candida* (Voglmayr and Riethmüller, 2006, Thines and spring, 2005).

4. TAXONOMIC STATUS

A. candida (AC) was first reported by Colmeiro in 1867 on brassica's 60-65 genera and 240-245 species (Choi et al., 2007). It is placed in Albuginaceae family with single genus 'Albugo'. Later four distinct lines were identified on different plant host and only S. strain

was infectious to Brassica (Voglmayr and Riethmüller, 2006; Thines and Spring, 2005). It was discovered during the eighteenth century but it was confused with *Uredo* and *Cystopus* spp. Later on, after extensive research it is regarded the only pathogen causing WR disease. Gmelin (1792) discovered and reported the first species of *Albugo* (Prev. *Aecidium candidum*) while previously it was categorized as sub-genus of the following genus. Later It was evaluated, categorized and differentiated between its species and regarded the *Albugo* as a separate genus (Riethmüller *et al.*, 2002). Initially, *Albuginaceae* was only family while later *Assteraceae* and *Brassicaceae* were deployed (Voglmayr and Riethmüller, 2006). Multiple distinctive lines or strains are present on the single similar plant family while some species are highly host specific to this genus. Mostly species of *Albugo* are parasitic to *Brassicaceae*. *A. Candida* has proverbial binomials like *cruciferarum*, *sphaericus*, *C. candidum*, *A. wasabiae*, *A. macrospora*, *A. lepidii*, *U. cheiranthi* and *U. Thlaspii*. This is biotrophic pathogen to *Brassicaceae* and WR infection was nearly common on cosmopolitan *Brassicaceae* (Choi *et al.*, 2006).

It was first placed in the *Proteomyceae* family by Gray and de-Bary shifted it to the *Peronosporaceae* family with common name *Cystopus*. After few years when the sexual stage of was discovered its family was changed to new family *Albuginaceae* for its proper placement but its monogeneric name was already being adjusted within the mildew group (Kirk *et al.* 2001; Dick, 2001). Then there was the need to place it in *albuginales* order. Its 50 species were reported infecting Brassica and crucifers host plants. Different characters are used to distinguish species like the structure of sporangia to construct identification marks for species. While other features like metabolism, chemistry or ultra-structural symmetry is implied for this purpose (Thines and Spring, 2005; Riethmüller *et al.*, 2002). Oospores are sexual spores observed by de Bary (1866). Asexually produced sporangia are formed by zoospore or rarely by developing germ tube. The mycelium is non-septate feeding intercellularly by producing knob like haustoria (Farr *et al.*, 2004; Meena *et al.*, 2014).

5. SYMPTOMOLOGY

Diseases symptoms are found at the foliar point of the host. These are white or off-white in color and surface of leaf look raised after infection. It would be pin size of various forms and sizes. The pathogen can permeate in the soil or stick with seed as a spore developed in the hypertrophied part. Sexual spores produced as germinating oospores are the source of primary infection (Saharan *et al.*, 2010). The fungus infection first appears on all the aerial parts of plants resulting in two types of infection like general/local or systemic. Local infection shows symptom as a cover of cream to white color chalk like blisters which develop into lesions. It causes cell enlargement (hypertrophy) and deterioration of infected plant part. Systemic infection appears as the formation of malformed meristem and inflorescence (racemes) commonly described as stag-head (Mishra *et al.*, 2009; Meena *et al.*, 2014).

Mode of survival and spread

A. candida (AC) is an obligate parasitic fungus. Two to three types of spores are produced and spread by wind and rain. Mycelium overwinters in infected material/debris and lives dormant in off season to resume its activity during new growth. The non-septate mycelium attack to get nutrients intercellularly through haustoria (Roland *et al.*, 2006; Verma, 2012). Reproduction takes place by sexual and asexual spores. Sporangia are produced on sporangiophore (asexual) while oospores are sexual spores. It survives in the soil and keeps itself alive on seed or in hypertrophied tissues. Primary infection is by means of Sexual spores while secondary infection/spread is caused by asexual spores like sporangia produced in diseased plant parts (Saharan, 2010). Disease prevalence after the attack is favored at 13-18°C temperature, 70-80% relative humidity (RH) and 3.8 to 4.5 km/h of wind velocity with recurrent raining at the field (Chattopadhyay *et al.*, 2011).

Mode of Action

Oospores are seed and air borne sexual spores when carried on seed, or overwintered in soil; serve as a prime source of primary inoculum. In resting stage, oospores germination is either due to the formation of 1-2 simple septated tubes or due to 50-55 zoospores released from aerial vesicles. Bursting of vesicle wall allows zoospores to be released in air or soil and germinate by germ tubes again on other plant (Verma, 2012). Mode of entry of fungus is through the host leaves 'stomata using knob like haustoria. There it develops more than twelve (12) hyphae per cell around mesophyll cells as a downward coil forming and penetrate the individual cells. Eventually intercellular spaces are filled by non-septic mycelial branches and fungal development take place. Maturing spores look short and club shape developed on sporangiophores having dense layer of mycelium and later show specific septation pattern unusual than earlier stages of vegetative mycelial growth. These sporangiophores are formed in closer to each other in the epidermis of the host. Several sporangia are developed on each sporangiophore but older starts to mature first. Studies investigations shows that not only mycelium but also the production of a large amount of sporangiophores and sporangia exert

pressure on the host epidermis leading it to burst. That's why leaves surface appeared whitish like a crust due to these sporangia (Saharanand Mehta, 2002).

Perennial weeds provide source of primary inoculum. Optimum temperature required for zoospores germination is 11–20°C and relative humidity 70% during primary infection. Secondary inoculum disperses through sporangial spore and air splashes serve to break the sporangia formed in mature pustules while presence of moisture on leaves helped in germination and infection. Thus, spores float in water for a short duration, produce germ tubes and move forward towards stomata to take entry. Another primary source of inoculum is mature hypertrophied plant at maturity stage providing space for oospore development (Goyal *et al.*, 1996).

Sometimes harvested seeds get mixed with pieces of malformed tissues having multiple oospores/zoospore or sporangiospore of the pathogen and fall on the ground. These infected seeds also become the source of pathogen infestation causing the disease. Mycelium developed on horse radish penetrates through infected crowns and lateral roots. But this mycelium is remained suppressed in off season but its growth is revived in new shoots during cropping season (Lakra and Saharan 1989). Over summered spores present in residues result in infection of *A. candida*. These spores were observed in naturally infected old leaves of *Brassica rapa* var. Toria (Lakra and Saharan, 1989; Goyal *et al.*, 1996; Saharan and Mehta, 2002).

6. RACES DEVELOPMENT AND VARIATION

There are multiple races of *A. candida* having different ability to cause disease. In a case study of *Brassica* different strains like 2, 3, 4, 5 and 6 are identified from *B. juncea*, *A. rusticana*, *C. bursa-pastoris*, *S. officinale*, and *R. islandica*. While race 7 and 8 is reported by Verma, Delwiche and Williams (1977) from *B. rapa* (Turnip) and *Brassica nigra*. Morphological forms and races of pathogen are easily differentiable in heterogeneous populations (Verma *et al.*, 1999). Geographical and climatic variations leave their imprints on it. There are nine specific races identified by Singh and Bhardwaj (1984) during field trials on 12 *Brassica* species of variety Toria (*B. rapa*). Different races infect different varieties like race 2 is infectious for *B. juncea*, *Brassica rapa* while race 3 attack on toria only. After the evaluation of crucifer's germplasm by Bhardwaj and Sud (1988), they reported 9 new races from brown Sarson (BSH-I), Toria (OK-I), *B. juncea* (Varuna) in India (Rimmer *et al.*, 2000). Different strains are collected from various places and evaluated to find variation and spread of multiple races in the area. Race 7 is further categorized into 7a and 7v isolates based on their virulence. Tower isolates 11-6 and 41-4 of *B. napus* are infectious for two species e.g. *B. rapa* and *B. juncea* and these are evolved after the cross of race 2 and race 7 (Rimmer *et al.*, 2000). Gupta and Saharan, (2002) evaluated different cultivars of *Brassica juncea* against *A. candida* and identified twenty (20) prominent subtypes and pathotype 2 & 17 of cultivar brown sarson specifically belong to *A. candida*. Pathotype (AC-2V) and (AC-2A) of *R. raphanistrum* and *B. juncea* are reported from Western Australia (Kaur *et al.*, 2008).

Variations in races and pathotype's of *A. candida* are documented from whole world. There are nine races reported from Australia, twenty from Britain, four from Canada, two from Germany, forty-nine from India, eight from Japan, eighteen from Rumania, and seven from the USA but their differentials need further verification to distinguish them. Different mechanisms are implied to study host differentials and pathogen interaction to evaluate racial variation like reproduction (oospores production especially on *B. juncea*), recombination, mutation, and heterokaryosis. Study of virulence range of *A. candida* is important for genetics. Standard nomenclature of such races was started by Saharan, (2010). Pathotypes of AC from *B. juncea* have multiple infection causing genes. Biotypes (AC-023), (AC-024), and (AC-17) attack only on three different hosts with limited virulence potential while AC (18, 21, 27, 29, and 30) have wide host range to infect due to higher level of potential virulent genes (Gupta and Saharan 2002).

Different isolates of AC imported from Australia were tested using complete internal transcribed spacer (ITS) rDNA nucleotide sequence and compared with isolates from Europe, Asia, and Australia. Most of the isolates are linked phylogenetically to Australian isolates. Few of them showed slight divergence with a distinct group of Western Australia native genotype. But mostly isolates of *Brassica* are genetically different from the prominent isolates analysed (Kaur *et al.*, 2011a, b).

7. BIOCHEMICAL CHANGES

A. candida (AC) alters the host biochemical system by infecting its mechanisms or machinery. Plant's defense system is activated by gene for gene interaction or recognition systems and produce virulence factors (Jones and Dangl, 2006). Oxidative burst triggers defense response in host by Producing Reactive Oxygen Species (ROS). ROS production initiates the hypersensitive action (a programmed cell death). This response limits access of fungus to water and food (Glazebrook, 2005). Although the synthesis and accumulation of reactive oxygen species (O_2 , O_2^- , OH^- , H_2O_2) is toxic but these are core product of metabolism. While their excess production due to infection result in oxidative stress in several plant tissues. The defense pathways require rapid induction of reactive oxygen species (ROS) and accumulation of phytoalexins (De Gara *et al.*, 2003; Agrios, 2005).

The ROS have superoxide anion ($O_2^{\cdot-}$) and hydrogen peroxide (H_2O_2) commonly interlinked to normal plant metabolic processes involving multiple antioxidant enzymes production like peroxidases, superoxide dismutase, catalase and ascorbate peroxidase during the pathogen attack (De Gara *et al.*, 2003). Among ROS, superoxide radicals are most damaging to cellular structures (De Gara *et al.*, 2003; Agrios, 2005). Biochemical changes are interlinked with resistance as a response of inducers like phytoalexins, lignin, and plant pathogenic proteins. Such inducers are developed through secondary xylem vessels in host (De Gara *et al.*, 2003). Proline alleviates the oxidative damage in lettuce by lowering lipid peroxidation and H_2O_2 content. After radiation exposure with endogenous Proline application, total-phenolic concentration and the antioxidant capacity of plant is enhanced. Additionally, exogenous application of Proline increases the levels of Gibberellic Acid, IAA, the concentrations of soluble sugars and organic acids and expressions of *PAL*, γ -TMT and *ProDH* genes while pre-treatment with exogenous Pro provides important contributions in regulating biochemical mechanism (Aksakal *et al.*, 2016).

Phenolic compounds are secondary metabolites which follow phenylpropanoids-flavonoids pathways (Latinize *et al.*, 2006). Secondary metabolites protect the plants from UV radiation and oxidants. They also act as defense and signal compounds. Plants cannot move like animals so plants cannot depend on physical mobility to escape from predators that's why plants have chemical defense mechanisms against their predators. Phenolic compounds play a significant role with the resistance of plants against the infective entities. Phenolic compounds serve as precursors to structural polymers such as lignin and have antimicrobial action. Enhanced peroxidases and PPO activity is found in resistant brassica plants after pathogen attack. In resistant cultivars, the activity of phenolic and o-phenols is higher resulting in high oxygenation of o-phenolic material to form more toxic substances for pathogens (Dussert *et al.*, 2003; Benson and Bremner, 2004).

Although, increased level of ROS is cytotoxic but its production is necessary to synchronize many prime physiological processes including, programmed cell death, hypertrophy and regulating oxidation-reduction sensitive signal transfer pathways. It causes cell damage, dis-mutations, and carcinogenesis. Antioxidants were produced to manipulate the damage caused by ROS in plant tissues as well as to regulate them. It was done by up-regulating anti-oxidative enzymes activity (Cho *et al.*, 2009). SOD, catalases, peroxidases, and GPX are necessary enzymes for all oxygen metabolizing cells. SOD is first layer of defense for the cyto-toxic effect of enhanced level of ROS. SOD converts molecular oxygen radical to hydrogen peroxide. The hydrogen peroxide is then regulated by catalase and a variety of peroxidases. Catalase play role in converting H_2O_2 into water and oxygen (Umasthun *et al.*, 2012).

Healthy and diseased *Brassica juncea* leaves were compared for Quantification analysis of peroxidase, invertases, ascorbic acid oxidase activity. Maximum Peroxidase and invertase concentration was reported in infected leaves in contrast to healthy ones. Amount of ascorbic acid oxidase production lowered with severity of infection (Singh *et al.*, 2011). Similarly, Enhanced temperature prominently increased the concentration of SOD (46%), POX (66.1%), and CAT (42%). CAT (Catalases) is protein of metabolite-system which prevents accumulation of H_2O_2 in the cells. Activity of catalase enzyme will be increases with rapid production of SOD in plants (Junmin and Jin, 2010). H_2O_2 function as to delimit the cell destruction by activating cell protective genes in surrounding cells. Enhanced level of H_2O_2 resulted in stimulating the resistance inducers in plants. However, it is down-regulated by CAT and POD, which prevented harmful effects of increased H_2O_2 in cells (Singh *et al.*, 1999).

POD plays a significant role in defense response of plant to against diverse infections. Peroxidase involves in production and break down of hydrogen peroxide. The induction of this enzyme occurs in case of both stimulus i.e. microbes; chemicals-agents and elicitors. its increase is linked with enhanced lipid per oxidation and decrease in membrane permeability (Kumar *et al.*, 2016). These enzymes serve as biochemical markers to develop resistant sources.

8. SOURCE OF RESISTANCE

In different countries *Brassica* and cruciferous germplasm was initially less resistant and there was need to identify it in field via screening. Later susceptible germplasm was used to develop resistant cultivars through mass selection (Saharan, 2010). In India, a screening experiment against WR pathogen was conducted in which forty-four genotypes out of which twenty-two Indian, twelve Australian, and ten Chinese were screened by manual inoculation under control condition. Four Chinese genotypes (CBJ-001) & one short continent genotype (J.R.4.9.) show resistance against WR pathogen (Caixaet *et al.*, 2007).

Various genotypes of *Brassica*, *B. carinata*, *B. napus* & *B. rapa* were evaluated in field conditions against white rust for five years. This long trial demonstrated that nine elite lines were resulted in disease incidence below 5% consistently for 3 to 4 years (Gaur *et al.*, 2015). Another experiment was designed on highly susceptible variety of *B. juncea* (RH-819) and its susceptibility was previously confirmed via two additional screening trials conducted under controlled conditions. Throughout the experiment, seeds were grown in hot water bag in plastic trays with 8 cells in optimum conditioned rooms balancing 12 to 19°C temp. with a 16hour light and optimum-intensity. In controlled environment variety RH-819 showed better growth and resulted in less exposed to disease (Li *et al.*, 2007b).

Research was designed in which twelve cultivars of brassica were evaluated against *Albugo candida* under field conditions. *Brassica juncea* and *Brassica napus*'s elite lines 44 S-01 and SPS-N7/26 were screened out which previously were declared highly resistant against white rust. Optimum conditions were provided for growth and allowing inoculums to build on plants. Established inoculums damaged its resistance and suggested that there is some change in genetics brought by the pathogen (Hina *et al.*, 2014). With the advancement of techniques and technology trend of screening turned to genetic studies to find resistant genes to exploit and modify them for the development of resistant cultivars.

Genetic study of host cultivars was carried out to find out the resistant genes against WR. Histological research revealed that resistance was controlled by a single dominant gene. It was regarded as a hypersensitive response in *Raphanus sativus* (*Caudatus*) cultivars as it was basically the manifestation of environmental conditions altering the minor genes and affecting the inoculums pressure tolerance reaction. The dominant resistant gene was named as "R" gene which later was described more precisely with the word of "AC-1" differentiated and reported from race 1 of *A. candida*. This discovery opened new horizons of finding different races and strains affecting different brassica hosts (Singh *et al.*, 1999).

Wang *et al.*, (2000) designed a screening trial to evaluate resistance in *Brassica napus* cultivars against blight and WR disease at different growth stages e.g. cotyledon stage. Few plants showed resistance against blighted disease and higher level of resistance against WR was also observed at successive growth stages. Similarly, Coelho and Monteiro, (2003) evaluated and compared *Brassica oleracea* lines at cotyledon stages and at adult stages and revealed that resistance is interlinked but type of resistance unknown. Doullah *et al.*, (2006) research findings revealed similar results from the screening of different cultivars of *Brassica rapa* against blight disease.

Whole plant bioassay of different *Brassica napus* cultivars was carried out in greenhouse conditions and transgenic lines were developed there. Resistance parameters like lesion size, minor disease intensity and stunted growth associated with *B. napus* was evaluated. Significant results were observed having ability to tolerate disease (Kanrar *et al.*, 2002). Rapid advancement in different techniques like tissue culture, protoplast fusion, embryo rescue, genetic engineering made it easy to transfer resistance genes/traits in different plants across the globe. Transgenic plants' resistant genes are over expressed against fungal pathogens which act as antifungal compounds e.g. Pathogenesis-related proteins. Their inhibitory activity was reported to be less efficient (Yun *et al.*, 1997).

It was reported that over-expressiveness of chitin hormone has ability to induce resistance by inhibiting the fungal growth ranged from 15-60% in trans-genic lines of brassica over non- transgenic control (Mondal *et al.*, 2008). It was studied that the pathogenesis related protein "osmotin". It was introduced in transgenic line of *Brassica juncea* which initiated the defence mechanism and induced tolerance against fungal attack. In addition to this osmotin also interfered with p53 and plant Cell Degrading Pathway (PCD) which assist plant to delay the symptoms appearance but could not provide adequate resistance (Reis *et al.*, 2011).

Two experiments conducted in which four different elite lines of *B. napus* were screened at different locations against WR and Alternaria. Fresh spore suspension was inoculated on brassica leaves and targeted pathogen development was observed. Two lines reported susceptible as severe chlorosis and necrosis was observed while other two lines showed resistant response significantly. Genetic study of the following lines was carried out by Subramanian and Bansal (2005) in University of Alberta during canola breeding programme. Genetic makeup was made resistant against blackleg while tolerance level of these lines was also recorded against leaf spot of Brassica (Sharma *et al.*, 2006). He applied carbonic anhydrase (CA) on brassica's line which were tolerant against *Alternaria* which resulted in satisfactory against the pathogen. He claimed that enhanced level of CA can increase the tolerance level of *B. napus* against *A. brassicae*. Another enzyme named Cinnamyl Alcohol Dehydrogenase (CAD) was involved in metabolic pathways and enhance tolerance level of *B. napus* lines against fungal pathogen after the exposure of 48hours (Rahman *et al.*, 2006). Silencing of Carbonic Anhydrase (CA) genes expression in tobacco leaves would hide the Pto mediated Hyper-sensitive reaction which will play major role in effectively activating the defence system in plants (Slaymake *et al.*, 2002).

Sanjay and Kumar, (2006) concluded that metabolites and external proteinoids can be provoked in Brassica plants by the action of benzothiadiazole treatment. They studied that management of brassica cv varuna with benzothiadiazole made alterations in the system of in-cellular proteins of total soluble phenols. The chronological increase in total soluble phenols was observed after BTH management. Mustard treated leaves with aqueous methanol extract of BTH produces new metabolite-phenol substance which was absent in untreated control. 12 soluble metabolic-proteins were present with MW ranged from 12.4 to 71.21 KDa collected from BTH treated mustard plants. Only few proteins P12.8, P1.2.7 and P.3.4.5 were present in a slight quantity in untreated control. It was observed that after 24 hours' treatment with BTH important proteins appeared with definite molecular weight of 33.0 and 33.7 representing towards their early initiation, while P-0 was most well-known protein which was appeared after 48 hours handling with BTH. It revealed that change in definite phenols and proteins with BTH treatment may develop resistance against mustard diseases.

It was reported that *Brassica alba*, *B. campestris*'s varieties including BSH-I, Champba, Gulivar, Sangam, SSK-I, TH-68), *B. carinata* HC-I, *B. juncea* D.I.R.1507, D.I.R.-522, ZEM-I), *B. napus*-Tower, , HNS-4, HNS-IO, Midas, Norin), *B. pekinensis*, *E. sativa* and *R. sativus* showed immunity against white rust in India. In another experiment, it was observed that varieties such as *B. alba*, HC-I, H-IIOA, PHR-I, DIR-1507, DIR-1522, TMS-50 EM-I, GS-7027, HNS-4, HNS- 10, Midas and Norin were also resistant against WR and downy mildew. It was demonstrated that HC-I and PCC-I of *B. carinata* and GSL-1501 of *B. napus* showed resistance against multiple diseases like white rust, Alternaria blight, powdery mildew (Saharan and Krishnia, 2001). It was also observed that genotypes EC-129 and Shevia were also free from white rust not only on leaves but also on inflorescences even under late sown conditions. These experiments were considered the best source of resistance genes and results of specie specific. Although first it was declared monogenic resistance with a complete over expression in *Brassica* which partial transferred to *B. juncea* lines by the selection of healthy plants in advance when separate generation sown in high disease pressure and their again and again crossing (Gupta *et al.*, 2002).

In a screening experiment the germplasm lines like GSL-1501, DOMO, B10YSR, Wester, GC 7027, RH 8539, PR 8805, DWRR 15, HNS 4, RN 248, JMM-W-7, EC-129126-1, GSL-1, HNS-3, DIR-1002, Midas, PC-3, Norin-14 & HC-1 were found resistant to white rust and downy mildew on the first observation. However above-mentioned starter lines the DOMO, B10YSR, Wester, RH-8539, DWRR-15, JMM-W-7; DIR-1002 & Norin-14 tend to show the susceptible reaction on last observation (Gupta *et al.*, 2002).

Another experiment of resistance against *pathogen* was conducted in which new and local potential cultivars were crossed. Results surprisingly demonstrated that immunity was controlled by an allelic & back revived via backcross breeding. Inheritance of resistance was confirmed by another research study of 153 cultivars conducted in open field conditions against disease of brassica revealed that gene Pi-15, was transferrable non-resistant type through backcrossing due single gene controllable (Jat and Saharan, 1999; Kole *et al.*, 2002).

WR resistant gene W.R.R.4. contain protein that gives wide range resistance in *A. thaliana* to *A. candida* races AC1to9 (Borhan *et al.*, 2008). Study of genetic changes and the possibility of increase type of WR resistance was evaluated in 7 specific host test of brassica cultivars *juncea* and *carinata* (Krishnia *et al.*, 2000). Such variations revealed that within family it was significant. The progenies testing of all 7 families, except resistant self-crossed and Susceptible self-crossed demonstrated prominent change for white Rust, disease score was reduced to 19% in WR. The maximum disease index (DSI) reported by progeny of susceptible self-crossing and minimum in resistant self-crossing test (Krishnia *et al.*, 2000; Borhan *et al.*, 2008).

Brassica plants defence was initiated while were isolated and amplified from host (Hulbert *et al.*, 2001). Genes should be expressed timely when needed to trigger defence during pathogenesis and its interactions, manifesting chlorophyll balances resulting in successful defence response (Borhan *et al.*, 2004).

9. MANAGEMENT APPROACHES

Different control measures are adopted to overcome WR. Cultural practices like crop rotation and weeds removal are also effective in reducing the inoculum pressure. Chemical control is one of the best, effective, rapid and robust methods to manage disease. Early reports were assumed to implement copper based fungicides like Bordeaux mixture which resulted in better control. Nissar *et al.*, (1990) designed an experiment which demonstrated that application of fungicides with weeds removal resulted in effectively controlling the disease. When Dithane M-45 (0.2%) and Dithane-Z78 were used as foliar spray one after other. Both fungicides helped in controlling disease and increasing the yield. Similar findings were reported by Morrall *et al.*, (1999) in which he demonstrated that crop rotation for two to three years with non-host crucifer's plants with two to three spraying of fungicides can reduce the disease incidence on canola crop. This will also reduce the effect of black leg pathogen effect on disease. It was also further evaluated by Johnston (2000) and he also recommended it to control the leaf spot diseases of brassica. The effect of number of sprays of Rovral 50 WP was evaluated by Hussain (1993) at Regional Agriculture Research station Bangladesh during 1991 to 1993 on mustard variety toria 7. Four plots were sprayed separately for one time, two times up to four times at ten days interval. Results revealed that greater yield was obtained by three sprays than four sprays of fungicides.

Seed treatment with bioagent like *Trichoderma harzianum* @ 10g/kg seed was evaluated by Rohilla *et al.*, (2001) followed by foliar spray of Red0ml-MZ-72 WP which effectively reduce pressure of *Alternaria brassicae* and *Albugo candida* in open field conditions. It helped not only in reducing the disease severity on brassica but also assisted in increasing the yield potential. Similar results were also demonstrated by Apron 35-SD when applied as a foliar spray mixed with Ridomil MZ-72 WP against WR and alternaria blight. Significant increase in yield was evident for their effectiveness. Native cauliflower variety Pusali was reported with 55% enhanced yield potential after the application of Rovral 50 WP by Ayub (2002) and commercial fungicides were found effective controlling many phytopathogens among bacterial and fungal foliar diseases (Johnston, 2002).

The spraying of Melathion-57 EC @ 1.55 ml/L was tested by Kodrathikhoda *et al.*, (2003) against insect infestation on brassica host in combination with the application of optimum fertilizer. It was later sprayed with iprodione for fungal pathogen. These chemicals were reported to assist each other to combat against fungal foliar diseases. Further it was claimed that Rovral 50 WP is efficient against *Alternaria* blight and WR Kohinor *et al.*, 2003). Similar findings were also documented by Hossain and Hosain, (2010) when they evaluated it on cauliflower against *Alternaria* blight (AB) when attacks inflorescence and causes reduced flowering. Flower drop was effectively prevented by foliar spraying of Rovral.

In another experiment Iprodione @ (2g/L) was evaluated by Hossain and Mian, (2005). In addition to fungicide he applied recommended doses of NPK fertilizer and treated the beds with micro-nutrients. This integrated management resulted in reduction of disease incidence up to 93% and enhanced production. Similarly, three chemical Captan, Mancozeb and Rovral were tested in open environment mixed with *Trichoderma harzianum* and *Pseudomonas fluorescence* against fungal pathogen infecting brassica plants. This was compared with methanol extract of medicinal plants like Ashoke, Eucalyptus and Calotropis. Results showed that medicinal plants significantly controlled the disease but chemicals also demonstrated comparable results in lessened the disease (Awasthi *et al.*, 2005).

A laboratory experiment was designed by Surviliene and Dambrauskiene, (2006) in which effect of different commercial fungicides (Amistar 250 SC, Folicur 250 EW, Signum 334 WG, and Zato 50 WG) with active ingredient was evaluated against *Alternaria* species on brassica. Colony growth was observed and measured using potato dextrose agar medium. Results showed that 25 to 90% colony growth was inhibited. Different concentrations and combinations of Bavistom, Topsin M and Ridomil MZ were evaluated and compared with other four commercial fungicides like Captaf, Indofil M-45, Indofil Z-78 and Thiram in vivo and in vitro conditions against brassica's diseases. Inoculum was applied prior to spraying of different of different concentrations like 50ppm, 100ppm, 150ppm and 200ppm. Ridomil MZ was found significantly effective controlling disease up to 32%. Similarly, the mixture of Baviston and Captaf reduced 25% disease. Application of these fungicide also supported in enhancing the yield potential up to ten-fold (Khan *et al.*, 2007). Seed treatment with Mancozeb followed by foliar application revealed its effectiveness in reducing the disease incidence (Mondal *et al.*, 2008).

Field experiment was performed by Rathi *et al.*, (2008) at Hisar, Haryana, India during 2007-08 and 2008-09 on brassica plants to analyze the impact of different biocontrol agents and fungicides with different combinations for seed treatment and foliar application. Effectiveness was recorded against *Alternaria* blight and WR on Indian mustard (*Brassica juncea*). Experimenting *Trichoderma* (10g/K) spraying of Redomil.MZ-72 WP containing active ingredient of 8% metalaxyl and 64% Mancozeb 2g after two-month of sowing: it significantly reduced the disease incidence. Incidence of WR was reduced up to 40%. Seed production was significantly increased up to 27% by this treatment. Apron 35 S (Metalaxyl 35%) was also found more effective than previous treatment. It was used for seed treatment 6g/Kg seed followed by foliar spray of RedomilMZ-72 WP 2g/L after 60 days interval after sowing. However, this combination controlled 65% and 40% WR and stag-heads disease respectively. Seed yield was significantly enhanced up to 38% by this application. In last combination Baviston (Carbendazim 50%) 2g/Kg and same foliar application of fungicide demonstrated almost similar results compared to previous treatments (Rathi *et al.*, 2008).

Frequent spraying of Dithiocarbonate was introduced to control the disease but it was less effective against stag head phase of disease. Bhatia and Gangopadhyay (2008) evaluated and reported that highest production was obtained by applying three times spraying of Ridoil-MZ @ 0.144% after infection and Apron-35 SO @ 2.1% after 50, 60 and 80 days interval. Less disease reduction was reported by the application of Mancozeb, captan and chlorothalonil while Tridemorph revealed toxic effects on the canola crop and foliar spraying of different doses of chemicals had no significant difference (Bhatia and Gangopadhyay, 2008). Acylalanine's application either foliar or soil drenching or seed dressing was found influential to control staghead phase of WR. Polyram @ 0.2% was evaluated on different varieties and reported effective in controlling WR on *Brassica rapa* var. sarson. Calixin @ was evaluated and recorded to be effective in managing the disease and in increasing crop production (Singh *et al.*, 2002).

Different chemicals in combination with different plant extracts against AB and WR disease on brassica leaves and pods were used (Sing *et al.*, 2011). It was reported that highest efficiency was recorded by Mancozeb against both diseases up to 64% and 19%. Similar results were obtained when it was combined with garlic extract up to 61%. *Brassica juncea* is one of the highly susceptible and easily invaded by *Alternaria brassicae* (*Alternaria* blight), *Albugo candida* (WR), *Erysiphe cruciferarum* (Powdery Mildew) and *Sclerotinia sclerotium* (*Sclerotinia* rot). Meena *et al.*, (2011) conducted several experiments at various locations for the management of such diseases during 2006 to 2009 in India. Foliar sprays with chemical fungicides alone and in combination with Eco-friendly products like *T. harzianum* and *Pseudomonas fluorescence* demonstrated less effective results against *Alternaria* blight but showed significantly superior and better results against WR and reduced the disease severity on leaves. Seed treatment of Bio-products also revealed effective results against WR pathogen suppression. Average yield was significantly improved by the following treatments (Meena *et al.*, 2011). In another experiment Meena *et al.*, (2011) studied chemicals like ZnSO₄, Borax, S, K and CaSO₄. Different plant

extracts like *Eucalyptus globosus* aqueous extract of leaf (50g/L) and galic (*Allium sativum*) bulb (20g/L) and Cow urine and bio-agents e.g. *T. harzianum* and *Pseudomonas fluorescence* were tested. Chemicals were proven to be very effective in controlling the Alternaria and WR pathogen. Other combinations also showed significant results with significant reduction of disease severity (Meena *et al.*, 2011. A field experiment was carried out to evaluate the efficacy of six fungicides to control white rust (*Albugo occidentalis*) of red amaranth (*Amaranthus* spp.) The fungicides were Sunvit @ 0.2%, Ridomil gold 68 WP (Chlorothalonil + Mefenoxam) @ 0.2%, Contaf 25 EC (Triazole) @ 0.1%, Orzim 50 WP (0.1%), Zoom 50 WP (0.1%), and X-tra care 300 EC (myclobutanil) % 0.05%. These were applied as foliar spray. The fungicides caused 8.18-70.28% reduction in severity in terms of percent disease index of white rust. On the contrary, the fungicides gave 22.31-110.50% increase in red amaranth fresh yield and 8.06-27.42% increase in 1000-seed weight. Among six fungicides tested, the most effective one to control white rust and to increase yield of red amaranth was Ridomil gold followed by Sunvit. Based on the findings of the present investigation Ridomil Gold 68 WP @ 0.2% foliar spray may be recommended to control white rust of red amaranth (Talkuder *et al.*, 2012).

10. FUTURE TRENDS IN RESEARCH

- Presence of oospores inside the seeds, and their possible importance in the survival of the pathogen needs further exploration.
- There is need to explore germinating oospores either simple or branched symmetry and morphology of germ tube. Single spore culture germinating from sporangia and from germinating oospores must be prepared and their pathogenicity compared.
- After screening lines for resistance against foliar infections, some select advanced lines must also be screened for production of white rust or stag heads.
- Identification of biological races of *A.candida* occurring on different Brassica crops in Pakistan needs a comprehensive study.
- Genetic studies are very important and biochemical or enzymatic study after regular interval of inoculation needs further extensive research.
- Quick remedy like application of fungicides is effective way to control the disease but the efficiency of different systemic and contact fungicides need to be explored

REFERENCE

1. Abada, K.A., Hilall, R. Mervat and S.H. Mostafa. 2008. Induced resistance against powdery mildew in cucumber. J. Biol. Chem. Environ. Sci. 3 (3): 45-56.
2. Abbas, J.S., F. Ullah, I.A. Khan, B.M. Khan and M. Iqbal. 2008. Molecular and biological assessment of Brassica napus and indigenous campestris species. Pak. J. Bot. 40(6): 2461-2469.
3. Abbas, J.S., F. Ullah, I.A. Khan, B.M. Khan, and M. Iqbal. 2009. Molecular analysis of genetic diversity in brassica species. Pak. J. Bot. 41(1): 167-176.
4. Abidi, T. and H. Pakniyat. 2010. Antioxidant enzyme changes in response to drought stress in ten cultivars of oilseed rape (*Brassica napus* L.). Czech J. Genet. Plant Breed. 46(1): 27-34.
5. Ahmad, M., Fautrier, A.G. and McNeil, D.L., 1997. Identification and genetic characterization of different resistance sources to Ascochyta blight within the genus Lens. Euphytica. 97: 311-315.
6. Aksakal, O., D. Tabay, A. Estringu, F.I. Aksakal, and N. Esim. 2017. Effect of proline on biochemical and molecular mechanisms in lettuce (*Lactuca sativa* L.) exposed to UV-B radiation. Photochem. Photobiol. Sci.
7. Ali, N. T., F. Ullah, M.A. Rabbani and Z.K. Shinwari. 2012. Genetic diversity in the locally collected brassica species of Pakistan based on microsatellite markers. Pak. J. Bot. 44(3): 1029-1035.
8. Armstrong, T. 2007. Molecular detection and pathology of the Oomycete *Albugo candida* (white rust) in threatened coastal cresses. DOC Research and Development Series 274. Department of Conservation, Wellington: 18.
9. Awasthi, M. and L.C. Rai. 2005. Toxicity of nickel, zinc, and cadmium to nitrate uptake in free and immobilized cells of *Scenedesmus quadricauda*. Ecotoxicol environ. Saf. 61(2): 268-272.
10. Bansal, V. K., J. P. Tewari, G. R. Stringam and M. R. Thiagarajah. 2005. Histological and inheritance studies of partial resistance in the *B. napus*-*A. candida* host-pathogen interaction. Plant Breed. 124: 27-32.
11. Benson, E.E. and D. Bremner. 2004. Oxidative stress in frozen plant: a free radical point of view. In Life in frozen state. Edited by F.B. Lane and E.E. Benson. CRC Press Inc., Boca Raton, Fla. pp. 256-269
12. Bhatia, J.N. and S. Gangopadhyay. 2008. Studies on chemical control white rust disease of mustard. Int. J. Pest Mana. 42(1): 61-65.
13. Borhan, M. H., E. B. Holub, J.L. Beynon, K. Rozwadowski and S. R. Rimmer, S. R. 2004. The Arabidopsis TIR-NB-LRR Gene RAC1 confers resistance to *Albugo candida* (white rust) and is dependent on EDS1 but not PAD4. Mol. Plant Microbe Intrac. 17:711-719.
14. Borhan, M. H., N. Gunn, A. Cooper, S. Gulden, M. Tor, S. R. Rimmer, and E. B. Holub. 2008. WRR4 encodes a TIR-NB-

- LRR protein that confers broad-spectrum white rust resistance in *Arabidopsis thaliana* to four physiological races of *Albugo candida*. *Mol. Plant-Microbe Interac.* 21: 757–768.
15. Bradford, M.M. 1976. A rapid and sensitive method for the quantitation of microgram quantities of protein utilizing the principle of protein-dye binding. *Anal. Biochem.*, 72: pp. 248–25.
 16. Chattopadhyay, C., A. Ranjana, Kumar, A. Meena, R.L.F. Karuna, N.V.K. Chakravarty, K. Ashok; G. Poonam, P. D. Meena and C. Shekhar. 2011. Epidemiology and development of forecasting models for White rust of *Brassica juncea* in India. *Arch. Phytopath. Pl. Prot* 44: 751–763.
 17. Chattopadhyay, C., R. Agrawal, A. Kumar, L.M. Bhar, P.D. Meena, R.L. Meena, S.A. Khan, A.K. Chattopadhyay, R.P. Awasthi, S.N. Singh, N.V.K. Chakravarty, A. Kumar, R.B. Singh, and C.K. Bhunia, 2005. Epidemiology and forecasting of *Alternaria* blight of oilseed *Brassica* in India – a case study. *J. Pl. Dis. Prot.* 112: 351–365
 18. Choi, Y. J., H.D. Shin, S. B. Hong and M. Thines. 2009. The host range of *Albugo candida* extends from Brassicaceae through Cleomaceae to Capparaceae. *Mycol. Prog.* 8: 329–335.
 19. Choi, Y. J., H.D. Shin, S. B. Hong S. Ploch and M. Thines. 2008. Evidence for uncharted biodiversity in the *A. candida* complex, with the description of a new species. *Mycol. Res.* 112: 1327–1334.
 20. Choi, Y. J., H.D. Shin, S. Ploch and M. Thines. 2011b. Three new phylogenetic lineages are the closest relatives of the widespread species *Albugo candida*. *Fungal Biol.* 115: 598–607.
 21. Choi, Y. J., M. J. Park, J.H. Park, and H.D. Shin. 2011a. White blister rust caused by *A. candida* on Oilseed rape in Korea. *Plant Pathol. J.* 27: 192.
 22. Choi, Y. J., S.B. Hong, and H.D. Shin. 2006. Genetic diversity within the *A. candida* complex (Peronosporales, Oomycota) inferred from phylogenetic analysis of ITS rDNA and COX2 mtDNA sequences. *Mol. Phylog. Evol.* 40: 400–409.
 23. Choi, Y.S., S.Y. Lee, I.C. Bang, D.S. Kim, Y.K. Nam. 2009. Genomic organization and mRNA expression of manganese superoxide dismutase (Mn-SOD) from *Hemibarbus mylodon* (Teleostei, Cypriniformes). *Immunol.* 27: 571–576.
 24. De Gara, L., M.C. de-Pinto and F. Tommasi. 2003. The antioxidant systems vis-a-vis reactive oxygen species during plant-pathogen interaction. *Plant Physiol. Biochem.* 41: 863–870.
 25. Dick, M. W. 2001. *Straminipilous Fungi*. Dordrecht: Kluwer.
 26. Dussert, S., N. Chabrillange, J.L. Montillet, J.P. Angel, F. Engelmann, and M. Noirod. 2003. Basis of coffee seed sensitivity to liquid nitrogen exposure: oxidative stress or imbibitional damage. *Physiol. Plant.* 119: 534–543.
 27. Fahey, J.W., A.T. Zalcman and P. Talalay. 2001. The chemical diversity and distribution of glucosinolates and isothiocyanates among plants. *Phytochemistry.* 56: 5–51.
 28. FAOSTAT Database. 2004. Food and Agriculture Organization of United Nations.
 29. Farr, D. F., A.Y. Rossman, M. E. Palm and E.B. McCray. 2004. Online fungal databases, systematic botany& mycology laboratory, ARS, USDA.
 30. Feyes, B. J., L.J. Moisan, M.A. Newman, and J.E. Parker. 2001. Direct interaction between the *Arabidopsis* disease resistance signaling proteins, EDS1 and PAD4. *The EMBO J.* 20: 5400–5411.
 31. Foyer, C. H. and G. Noctor. 2000. Oxygen processing in photosynthesis: regulation and signaling. *New Phytologist*, 146 (112): 359–388. doi:10.1046/j.1469-8137.2000.00667.x.
 32. Freeman, L. J., A. Lomas, N. Hodson, M. J. Sherratt, K. T. Mellody and A. S. Weiss. 2005. Fibulin-5 interacts with fibrillin-1 molecules and microfibrils. *Biochem J* 388:1–5.
 33. Garcia, P. C., R. M. Rivero, J. M. Ruiz and L. Romero. 2003. The role of fungicides in the physiology of higher plants: Implications for defense responses. *The Bot. Rev.* 69(2):162–172.
 34. Gaur, R.B., R.N. Sharma and R.P. Meena. 2016. Multiple disease resistance in different *Brassica* genotypes. *J. Oil. Bra.* 7 (1): 98–105.
 35. Glazebrook, J. 2005. Contrasting mechanisms of defense against biotrophic and necrotrophic pathogens. *Annual Review of Phytopathology*, 43: 205–227.
 36. Gmelin, J. F. 1792. *Systema Naturae*. 2. G. E. Beer, Leipzig.
 37. Godika, S., and A. K Pathak. 2005. Control of white rust and *Alternaria* blight diseases of mustard by foliar sprays of Ridomil. *Pestology.* 29: 9–10.
 38. Goyal, A., D. P. Norton, J. D. Budai, M. Paranthaman, E. D. Specht, D.M. Kroeger, and D.F. Lee, 1996. High critical current density superconducting tapes by epitaxial deposition of YBa₂Cu₃O_x thick films on biaxially textured metals. *Appl. Phys. Lett.* 69(12): 1795–1797.
 39. Gupta, K. and G.S. Saharan. 2002. Identification of pathotypes of *Albugo candida* with stable characteristic symptoms on Indian mustard. *J. Mycol. Plant Pathol.* 32: 46–51.
 40. Gupta, K., D. Prem, M.S. Negi and A. Agnihotri. 2004. An efficient tool to screen interspecific F1 hybrids in *Brassicaceae*. In *Proceedings: 4th International Crop Science Congress*, 26th Sept - 1st Oct. 2004, Brisbane, Australia.
 41. Hagemeier, J., B. Schneider, N. Oldham and K. Hahlbrock. 2001. Accumulation of soluble and wall-wound indolic

- metabolites in *Arabidopsis thaliana* leaves infected with virulent or avirulent *Pseudomonas syringae* pathovar tomato strains. *Proceedings of the National Academy of Sciences, USA*. 98: 753–758.
42. Hahlbrock, K., P. Bednarek, L. Ciokowski, B. amberger, A. Heise, H. Liedgens, E. Logemann, T. Nurnberger, E.Schmelzer, I.E.Somssich and J. Tan. 2003. Non-self-recognition, transcriptional reprogramming, and secondary metabolite accumulation during plant/pathogen interaction. *Proceedings of the National Academy of Sciences, USA*. 100: 14569–14576.
 43. Hameed, A., N. Iqbal and S. A. Malik. 2014. Effect of D-mannose on antioxidant defense and oxidative processes in etiolated wheat coleoptiles. *Acta Physiol Plant*. 36: 161–167.
 44. Hammerschmidt, R. 2005. Phenols and plant-pathogen interactions: The saga continues. *Physiol Mol Plant Pathol.*, 66: 77–78.
 45. Hina N.A., R. Perveen, S. Chohan, G. Yasmeen, M. A. Mehmood, W. Hussain. 2014. Screening of canola germplasm against *Albugo candida* and its epidemiological studies. *Pak. J. Phytopathol*. 26 (02): 169–173.
 46. Hossain, M. S., and M. M. Hossain. 2010. Effect of *Alternaria* blight on the seed yield of cauliflower (*Brassica oleracea* L.). *Bangladesh J. Agric. Res.* 35(3): 381–385.
 47. Huber, L.S., R. Hoffmann-Ribani and D.B. Rodriguez-Amaya. 2009. Quantitative variation in Brazilian vegetable sources of flavonols and flavones. *Food Chem*. 113: 1278–1282.
 48. Hutcheson, S. W. 1998. Current concepts of active defense in plants. *Annu. Rev. Phytopathol*. 36: 59–90.
 49. Ibrahim, M. H., and H. Z. Jaafar. 2013. Absciscic acid induced changes in production of primary and secondary metabolites, photosynthetic capacity, antioxidant capability, antioxidant enzymes and lipoygenase inhibitory activity of *Orthosiphon stamineus* Benth. *Molecules*. 18(7): 7957–7976.
 50. Jahangir, M., H.K. Kim, Y.H. Choi and R. Verpoorte. 2009. Health-affecting compounds in Brassicaceae. *Comp. Rev. Food Sci. Food Saf.* 8: 31–43
 51. Jat, R. R., and Saharan, G. S. 1999. Inheritance of resistance in interspecific crosses between Indian mustard [*Brassica juncea* (L.) Czern. & Coss.] and rape (*B.napus* L.) to *Albugo candida* (Pers. Ex. Hook). *Indan Phytopathol*. 52: 319.
 52. Jones, J. D., and J.L. Dangl. 2006. The plant immune system. *Nature*, 444: 323–329.
 53. Junmin, L. and Z. Jin. 2010. Potential allelopathic effects of *Mikania micrantha* on the seed germination and seedling growth of *Coix lacryma-jobi*. *Weed Biol. Manag.* 10: 194–201.
 54. Kaur, P., K. Sivasithamparam and M.J. Barbetti. 2008. Pathogenic behaviour of strains of *Albugo candida* from *Brassica juncea* (Indian mustard) and *Raphanus raphanistrum* (wild radish) in Western Australia. *Aust. Plant Pathol*. 37: 353–356.
 55. Kaur, P., R. Jost, K. Sivasithamparam and M.J. Barbetti. 2011a. Proteome analysis of the *A. candida*-*B. juncea* pathosystem reveals that the timing of the expression of defence-related genes is a crucial determinant of pathogenesis. *J. Exp. Bot.* 62: 1285–1298.
 56. Kaur, P., R. Jost, K. Sivasithamparam and M.J. Barbetti. 2011b. Proteome analysis of the *A. candida*-*B. juncea* pathosystem reveals that the timing of the expression of defence-related genes is a crucial determinant of pathogenesis. *J. Exp. Bot.* 62: 1285–1298.
 57. Kaur, P., R. Jost, K. Sivasithamparam, H. Li and M.J. Barbetti. 2010. Host-pathogen interactions in the mustard white rust pathosystem: Protein expression profiling. *Phytopathol*. 100: S60.
 58. Khan, Z., Kim, Y. H., Kim, S. G. and Kim, H. W. 2007. Observation of the suppression of root-knot nematode (*Meloidogyne arenaria*) on tomato by incorporation of cyanobacteria power (*Oscillatoria chlorina*) into potting filed soil. *Bioresour. Technol.* 98:69–73.
 59. Kirk, P.M., P.F. Cannon, J.C. David and J.A. Stalpers. 2001. *Ainsworth & Bisby's Dictionary of the Fungi* 9th ed. Wallingford: CAB International.
 60. Kohinoor, H., S. Kodratikhoda, and I. H Mian. (2003). Foliar spray of fungicides and botanicals to control *Alternaria* blight of cauliflower seed crop. *Bangladesh J. Plant Pathol.* 19: 63–67.
 61. Kole, C., P. H. Williams, S. R. Rimmer, and T. C. Sborn. 2002. Linkage mapping of genes controlling resistance to white rust (*A. candida*) in *B. rapa* (syn.campestris) and comparative mapping to *B. napus* and *A. thaliana*. *Genome*. 45: 22–27.
 62. Kombrink, E. and E. Schmelzer. 2001. The hypersensitive response and its role in local and systemic disease resistance. *European J. Plant Pathol*. 107: 69–78
 63. Krishnia, S. K., G.S. Saharan and D. Singh. 2000. Genetic variation for multiple disease resistance in the families of interspecific cross of *Brassica juncea* x *B. carinata*. *Cruciferae Newsletter*. 22: 51–53.
 64. Kumar, D., N. Maurya, Y. K. Bharati, A. Kumar, K. Kumar, K. Srivastava, G. Chand, C. Kushawa, S. K. Singh, R.K. Mishra and A. Kumar. 2014. *Alternaria* blight of oilseed Brassicas: A comprehensive overview. *African J. Microbiol. Res.* 8(30): 2816–2829.
 65. Kumar, N., S. C. Shankhdhar and D. Shankhdhar. 2016. Impact of elevated temperature on antioxidant activity and

- membrane stability in different genotypes of rice (*Oryza sativa* L.). *Indian J. Plant Physiol.* 21(1):37–43.
66. Kuzniak, E. and M. Sklodowska. 2001. Ascorbate, glutathione and related enzymes in chloroplasts of tomato leaves infected by *Botrytis cinerea*. *Plant Sci.* 160: 723–31.
 67. Lakra, B. S. and g. S. Saharan. 1989. Sources of resistance and effective screening techniques in Brassica Albugo system. *Indian Phytopathol.* 42: 293. (Abstract.)
 68. Lebeda, A., D. Jancov and L. Luhova. 1999. Enzymes in fungal plant pathogenesis. *Phyton (Horn, Austria).* 39(3): 51–56.
 69. Lebeda, A., L. Luhova, M. Sedlarova, and D. Jancova. 2001. The role of enzymes in plant fungal pathogens interactions. *J. Plant Dis. Prot.* 108: 89–111.
 70. Leonard, G., and N. Reigner. 2006. How the use of fungicides has benefited US agriculture, *Outlooks on Pest Management*, CropLife Foundation, Washington, DC, U.S.A.
 71. Levine, M. P., L. Smolak, A. F. Moodey, M. D. Shuman and L. D. Hessen. 1994. Normative developmental challenges and dieting and eating disturbances in middle school girls. *Int.J. Eat. Disor.* 15: 11–20.
 72. Li, C. X., K. Sivasithamparam, G. Walton, P. Salisbury, W. Burton, S.S. Banga, C. Chattopadhyay, A. Kumar, R. Singh, D. Singh, A. Agnohotri, S. Y. Liu, Y.C. Li, T. D. Fu, Y. F. Wang, and M.J. Barbetti. 2007. Expression and relationships of resistance to white rust (*Albugo candida*) at cotyledonary, seedling, and flowering stages in Brassica juncea germplasm from Australia, China, and India. *Australian J. Agric. Res.* 58: 259–264.
 73. Liu, N., Z. Lin, L. Guan, G. Gaughan and G. Lin. 2014. Antioxidant enzymes regulate reactive oxygen species during pod elongation in *Pisum sativum* and *Brassica chinensis*. *PLoS one.* 9(2): e87588.
 74. Lo Y.Y.C., J.M.S Wong, and T.F. Cruz. 2006. Reactive oxygen species mediate cytokine activation of c-Jun NH2- terminal kinases. *J. Biol. Chem.* 271: 15703–15707.
 75. Luck, J. E., G. J. Lawrence, P.N. Dodds, K. W. Shepherd and J.G. Ellis. 2000. Regions outside of the leucine-rich repeats of flax rust resistance proteins play a role in specificity determination. *The Plant cell.* 12:1367–1377.
 76. Mahdy, A. M. M., El-Mageed, A., Hafez, M. A and Ahmed, G. A. 2006. Using alternatives to control cucumber powdery mildew under green-and commercial protected-house conditions. *Fayoum J. Agric. Res. Dev.* 20: 121–138.
 77. Meena, P.D., P.R. Verma, G.S. Saharan, and M. H. Borhan. 2014. Historical perspectives of white rust caused by *Albugo candida* in Oilseed Brassica. *J. Oil. Bras.* 5: 1–41.
 78. Meena, S. S., K. S. Brar, J. S. Chauhan, P. D. Meena, P. S. Sandhu, R. P. Awasthi, A. S. Rath, Kumar, Ashok, J. C. Gupta, and S. J. Kolte. 2011. GGE Biplot analysis of Brassica genotypes for white rust disease severity under aided epiphytotic conditions in India (pp. 1201–1204). *Prague: Proceedings of 13th International Rapeseed Congress*
 79. Mhamdi, A., J. Hager, S. Chaouch, G. Queval, Y. Han, L. Taconnat, P. Saindrenan, H. Gouia, B. E. Issakidis, J. P. Renou and G. Noctor. 2010. Arabidopsis glutathione reductase 1 plays a crucial role in leaf responses to intracellular hydrogen peroxide and in ensuring appropriate gene expression through both salicylic acid and jasmonic acid signaling pathways. *Plant Physiology* 2010; 153: 1144–1160.
 80. Mishra, K.K., S.J. Kolti, N.I. Nishaat and R.P. Awasti. 2009. Pathological and biochemical changes in Brassica juncea (Mustard) affected with *Albugo candida* (white rust). *J. Pl. Path.* 58: 80–86.
 81. Mohammadi, M. and H. Kazemi. 2002. Changes in peroxidase and polyphenol activity in susceptible and resistant wheat heads inoculated with *Fusarium graminearum* and induced resistance. *Pl. Dis.* 162(4): 491–498
 82. Moieni, Z. K., G. Karimzadeh and M. Sharifi. 2013. Evaluation of total soluble protein and antioxidant activities in two spring cultivars of canola (*Brassica napus* L.) in response to low temperature. *Intl. J. Agri. Crop Sci.* 5 (4): 401–409.
 83. Mondal, G., T. K. Walli and A. K. Patra, 2008. In vitro and in sacco ruminal protein degradability of common Indian feed ingredients. *Livest. Res. Rural Dev.* 20 (4): 63.
 84. Morrall, R. A. A., D. A. Kaminski and L. A. Kaminski. 1999. The 1996–98 western Canada disease survey; Do agronomic practices affect disease or vice-versa?. *Proceedings of the 10th International Rapeseed Congress.* Canberra, Australia.
 85. Muhanna, A.S. and Naglaa. 2006. Pathological studies on root-rot and vine decline of cantaloupe in Egypt. *Ph.D. Thesis, Fac. Agric., Cairo Univ.* 218.
 86. Mustafa, H.S.B., E. Hasan, T. Mahmood, M. Aftab, F. Saddique and H. Rehman. 2017. Quantitative and qualitative evaluation of rapeseed (*Brassica napus* L.) genotypes for the development of high yielding canola quality cultivars. *Discovery.* 53(259), 380–387.
 87. Nissar A.A., M. W. Khan, and A. Muheet. 1990. Evaluation of some fungicides for seed treatment and foliar application in management of damping-off of seedlings and blight of rapeseed caused by *Alternaria brassicae*. *Mycopathologia* 110(30): 163–167.
 88. Orober, M., J. Siegrist and H. Buchenaue. 2002. Mechanisms of phosphate-induced disease resistance in cucumber. *European J. Plant Pathol.* 108(4): 345–353.

89. Pakistan Oilseed Development Board (PODB), Economic Survey of Pakistan. 2014-15, Federal Bureau of Statistics, MINFAL, Islamabad.
90. Pedras, M. S. C., Q. A. Zheng and V.K. Sarma-Mamillapalle. 2007b. The phytoalexins from Brassicaceae: Structure, biological activity, synthesis and biosynthesis. *Nat. Product Commun.* 2: 319–330.
91. Pedras, M. S. C., Q.A. Zheng, R. S. Gadagi and S.R. Rimmer. 2008. Phytoalexins and polar metabolites from the oilseeds canola and rapeseed: differential metabolic responses to the biotroph *Albugo candida* and to abiotic stress. *Phytochemistry*. 69: 894–910.
92. Pnueli, L., H. Liang, M. Rozenberg and R. Mittler. 2003. Growth suppression, altered stomatal responses, and augmented induction of heat shock proteins in cytosolic ascorbate peroxidase (Apx1)-deficient *Arabidopsis* plants. *Plant J.* 34(2): 187–203.
93. Pruthi, V., H. K. L. Chawla and G. S. Saharan. 2001. *Albugo candida* induced changes in phenolics and glucosinolates in leaves of resistant and susceptible cultivars of *Brassica juncea*. *Cruciferae Newsletter*. 23: 61–62.
94. Przybylski, R. and T. Mag. 2002. Canola/rapeseed oil. In: *Vegetable oils in Food Technology: composition, properties and uses* (F.D. Gunstone Ed.). Blackwell Publishing, CRC Press.
95. Quiroga, M., C. Guerrero, A.B. Miguel, B. Araceli, A. Irida, M. Medina, F. J. Alonso, S. M. de Forchetti, H. Tigier and V. Valpuesta. 2000. A Tomato Peroxidase Involved in the Synthesis of Lignin and Suberin. *Plant. Physiol.* 122: 1119–1127.
96. Rahman, I., Kode, A., and S. K. Biswas. 2006. Assay for quantitative determination of glutathione and glutathione disulfide levels using enzymatic recycling method. *Nature proto.* 1(6): 3159–3165.
97. Ranjan, M.S., P.C. Koley, M. Dasgupta and A. Mukherjee. 2009. Changes in Phenolics, Polyphenol Oxidase and its Isoenzyme Patterns in Relation to Resistance in Taro against *Phytophthora colocasiae*. *J. Phytopathol* 157:145–153. doi: 10.1111/j.1439-0434.2008.01458.x
98. Rashid, M.M.U., M.R. Hossain, M.N. Islam, M. Kamal, and M.Yusuf. 2014. Evaluation of cytotoxic and thrombolytic activities of methanolic extract of the flowers of *Sida acuta*. *Bull. Pharm. Res.* 4(3):108–111.
99. Rathi, A.S. and D. Singh. 2009. Integrated management of *Alternaria* blight and white rust in Indian mustard. *Proceedings of 16th Australian weed conference*.
100. Reis, P. A., G. L. Rosado, L. A. Silva, L. C. Oliveira, L. B. Oliveira, M. D. Costa, and E. P. Fontes. 2011. The binding protein BiP attenuates stress-induced cell death in soybean via modulation of the N-rich protein-mediated signaling pathway. *Plant physiol.* 157(4): 1853–1865.
101. Riethmuller, A., H. Voglmayr, M. Goker, M. Weis, and F. Oberwinkler. 2002. Phylogenetic relationships of the downy mildews (Peronosporales) and related groups based on nuclear large subunit ribosomal DNA sequences. *Mycologia*. 94, 834–849.
102. Rimmer, S. R., S. Mathur and C.R. Wu. 2000. Virulence of isolates of *Albugo candida* from western Canada to Brassica species. *Canadian J. Plant Pathol.* 22: 229–235.
103. Rohilla, R., R. L. Singh, U. S. Singh, R. P. Singh, E. Duveiller and H. B. Singh. 2001. Recent advances in the management of plant disease using chemicals (No. CIS-3524. CIMMYT.).
104. Rolland, F., E. Baena-Gonzalez, and J. Sheen. 2006. Sugar sensing and signaling in plants: conserved and novel mechanisms. *Ann. Review Plant Biol.* 57: 675–709.
105. Rossetto, M.R.M., T.M. Shiga, F. Vianello and G.P.P. Lima. 2013. Analysis of total glucosinolates and chromatographically purified benzyl glucosinolate in organic and conventional vegetables. *Food Sci. Technol.* 50: 247–252.
106. Sadiq, M., N. A. Akram And M. Ashraf. 2017. Foliar applications of alpha-tocopherol improves composition of fresh pods of *Vignaradiata* subjected to water deficiency. *Turk. J. Bot.* 41:1–9.
107. Saharan, G. S. 2010. Analysis of genetic diversity in *Albugo-crucifer* system. *J. Mycol. Plant Pathol.* 40: 1–13.
108. Saharan, G. S., and N. Mehta. 2002. Fungal diseases of rapeseed-mustard. In V. K. Gupta and Y. S. Paul (Eds.), *Diseases of field crops*. 193–228. New Delhi: Indus Pub. Co.
109. Saharan, G. S., and S. K. Krishnia. 2001. Multiple disease resistance in rapeseed and mustard. In: S. Nagarajan and D. P. Singh. (Eds.) *Role of resistance in intensive agriculture* (pp. 98–108). New Delhi: Kalyani.
110. Sanjay, G., and A. Kumar. 2006. Qualitative profiling of phenols and extracellular proteins induced in mustard (*Brassica juncea*) in response to benzothiadiazole treatment. *J. Cell Mol. Biol.* 5: 51–56.
111. SAS Institute, 1990. *SAS/STAT Users Guide Version 6*. SAS Institute, Cary, NC, USA.
112. Shahid, M., M. M. Khan, A. Hameed, M. Ashraf and A. Jamil. 2012. Antioxidant enzymes and inorganic elements in seeds and leaves of four potential medicinal plants from Pakistan
113. Shahzadi, T., F.K. Ahmad, F. Zafar, A. Ismail, E. Amin and S. Riaz. 2015. An Overview of Brassica Species for Crop Improvement. *American-Eurasian J. Agric. Environ. Sci.* 15 (8): 1568–1573.
114. Sharma, A. K. and S. K. Sharma. 1998. Peroxidase and polyphenol oxidase activity changes in relation to leaf rust of wheat. *J. Maharashtra Agric. Uni.* 22: 286–291.

115. Sharma, R. L., B. P. Singh, M. P. Thakur, and K. P. Verma. 2002. Chemical management of linseed wilt caused by *Fusarium oxysporum* f. sp. lini. *Annals Plant Prot. Sci.* 10(2): 365-410.
116. Singh Y., D.V. Rao, and A. Batra. 2011. Enzyme activity changes in *Brassica juncea* (L.) Czern.& Coss. In response to *Albugo candida* Kuntz (Pers.). *J. Chem. Pharm. Res.* 3(3):18-24.
117. Singh, B. and K. Usha. 2003. Salicylic acid induced physiological and biochemical changes in wheat seedlings under water stress. *Plant Growth Regul.* 39: 137-141.
118. Singh, B. M. and C. L. Bhardwaj. 1984. Physiologic races of *Albugo candida* on crucifers in Himachal Pradesh. *Indian J. Mycol. Plant Pathol.* 14, 25 (Abstract).
119. Singh, H. V. 2005. Biochemical changes in *Brassica juncea* cv. Varuna due to *Albugo candida* infection. *Pl. Di. Res.* 20: 167-168.
120. Singh, U. S., N.I. Nashaat, K.J. Doughty and R.P. Awasthi. 2002. Altered phenotypic response to *Peronospora parasitica* in *Brassica juncea* seedlings following prior inoculation with an avirulent or virulent isolate of *Albugo candida*. *European J Plant Pathol.* 108: 555-564
121. Singh, Y., R. S. Jamwal, N. K. Pathania and P. Kalia. 1997. Inheritance of biochemical traits in relation to buckeye fruit rot (*Phytophthora nicotianae* var. *parasitica*) in tomato. *Indian J. Hill Farm.* 10: 10-15.
122. Slaymaker, D. H., D. A. Navarre, D. Clark, O. del Pozo, G.B. Martin, and D. F. Klessig. 2002. The tobacco salicylic acid-binding protein 3 (SABP3) is the chloroplast carbonic anhydrase, which exhibits antioxidant activity and plays a role in the hypersensitive defense response. *Proceedings of the National Academy of Sciences*, 99(18): 11640-11645.
123. Steel, R. G. D., J. H. Torrie and D. A. Dickey. 1997. Principles and procedures of statistics. A biometrical approach. 3rd Edit. McGraw Hill Pub. Co., New York.
124. Subramanian, B., V. K. Bansal and N. N. Kav. 2005. Proteome-level investigation of *Brassica carinata*-derived resistance to *Leptosphaeria maculans*. *J. Agric. Food Chem.* 53 (2): 313-324.
125. Sullivan, M. J., J. P. Damicone, and M. E. Payton. 2002. The effects of temperature and wetness period on the development of spinach white rust. *Plant Dis.* 86: 753-758
126. Tan, J. W., P. Bednarek, J. K. Liu, B. Schneider, A. Svatos and K. Hahlbrock. 2004. Universally occurring phenylpropanoid and species-species indolic metabolites in infected and uninfected *Arabidopsis thaliana* roots and leaves. *Phytochemistry*. 65: 691-699.
127. Thines, A. and O. Spring. 2005. A revision of *Albugo* (Chromista, Peronosporomycetes). *Mycotaxon*. 92:443-458
128. Tirmali, A.M. and S.J. Kolte. 2013. Effect of pre- and post-inoculation application of amino acids on induction of resistance, peroxidase activity against *Albugo candida* (Pers) Kuntze in Indian mustard [*Brassica juncea* (L.) Czern & Coss.]. *Journal of Oilseed Brassica*, 4(1): 25-32.
129. Trimali, A. M. and Kolte, S. J. 2012. Induction of host resistance in mustard with non-conventional chemicals against white rust (*Albugo candida*). *J. Plant Dis. Sci.* 7(1): 27-31.
130. Umasuthan, N., S. Bathige, S.R. Kasthuri, L. Youngdeuk, W. Ilson, Y.C. Cheol, P. Hae-Chul and L. Jehee. 2012. A manganese superoxide dismutase (MnSOD) from *Ruditapes philippinarum*: Comparative structural- and expressional-analysis with copper/zinc superoxide dismutase (Cu/ZnSOD) and biochemical analysis of its antioxidant activities. *Fish Shellfish Immunolo.* 1-13.
131. Velikova, V., I. Yordanov and A. Edreva. 2000. Oxidative stress and some antioxidant systems in acid rain-treated bean plants: Protective roles of exogenous polyamines. *Plant Sci.* 151: 59-66.
132. Verma, P. R., G. S. Saharan, A. M. Bartaria and A. Shivpuri. 1999. Biological races of *Albugo candida* on *Brassica juncea* and *B. rapa* var. Toria in India. *J. Mycol. Plant Pathol.* 29: 75-82.
133. Verma, P.R. 2012. White rust of crucifers: An overview of research progress, Special Lecture during 1st National Brassica Conference. *J. Oilseed Bras.* 3(2): 78-87.
134. Voglmayr, H., and A. Riethmüller. 2006. Phylogenetic relationships of *Albugo* species (white blister rusts) based on LSU rDNA sequence and oospore data. *Mycol. Res.* 110: 75-85.
135. Wang, S. H., Z. M. Yang, H. Yang, B. Lu, S. Q. Li, and Y. P. Lu. 2004. Copper-induced stress and antioxidative responses in roots of *Brassica juncea* L. *Bot. Bulletin Academia Sinica*, 45.
136. Warwick, S.I, R.K. Guge, T. Mc-Donalad and K.C. Falk. 2006. Genetic variation of Ethiopian mustard (*Brassica carinata* A. Braun) germplasm in western Canada. *Genet. Resour. Crop Evol.* 53: 297-312.
137. Wulff, B. B., C.M. Thomas, M. Smoker, M. Grant and J. D. Jones. 2001. Domain swapping and gene shuffling identify sequences required for induction of an Avr-dependent hypersensitive response by the tomato Cf- 4 and Cf-9 proteins. *The Plant Cell.* 13: 255-272.
138. Yao Q.L, F.B. Chen, P. Fang, G.F. Zhou. 2012. Genetic diversity of Chinese vegetable mustard (*Brassica juncea* Coss.) landraces based on SSR data. *Biochem. Syst. Ecol.* 45: 41-48.
139. Young, N. D. 2000. The genetic architecture of resistance. *Cur. Opi. Pl. Biol.* 3: 285-290.
140. Yun, D. J., R. A. Bressan and P.M. Hasegawa. 1997. Plant antifungal proteins. *Pl. Breed Reviews.* 14: 39-88.

141. Zavareh, A.H.J., A. S. Tehrani and M. Mohammad. 2004. Effects of Acibenzolar-S-methyl on the specific activities of peroxidase, chitinase and phenylalanine ammonia-lyase and phenolic content of host leaves in cucumber powdery mildew interaction. *Commun. Agric. Appl. Biol. Sci.* 69(4): 555-563.